PUBLIC HEALTH

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INTRODUCTION

Operating the proposed East Altamont Energy Center (EAEC) would produce combustion products and other toxics to which the general public and workers might be exposed. Such exposures can produce specific health symptoms in humans and are the focus of federal and state requirements for specific technological and operational controls. The issue of possible worker exposure is addressed in the **Worker Safety and Fire Protection** section while the health significance of exposure to the project-related electric and magnetic fields (EMF) is addressed in the **Transmission Line Safety and Nuisance** section. Potential impacts from waste generation and disposal are discussed in the **Waste Management** section.

The air pollutants of specific concern for EAEC and similar gas-fired facilities are categorized as criteria pollutants and non-criteria pollutants. The non-criteria pollutants are also known as air toxics or toxic air contaminants (TACs) to reflect the nature of their biological interactions. The criteria pollutants differ from the air toxics in that the former have specific air quality standards, which were established to protect against significant health impacts in humans. The health impacts of criteria pollutants are discussed in **Public Health: Attachment A**, while the potential for air quality violations is addressed in the **Air Quality** section. When a project is proposed for an area with violations of the air quality standards, specific mitigation might be necessary to prevent significant additions to existing levels of the pollutants involved. Since this project is proposed for an air basin in violation of specific air quality standards as noted by the applicant (EAEC 2001a, pages 8.1-5 through 8.1-11, and pages 8.1-68 through 8.1-72), and discussed in the **Air Quality** section, specific mitigation is recommended in that section.

The purpose of this **Public Health** analysis is to determine if toxic emissions from the proposed EAEC would have the potential to cause significant adverse public health impacts or to violate standards set for the protection of public health. If potentially significant health impacts are identified, staff will evaluate mitigation measures to reduce such impacts to levels of insignificance.

LAWS ORDINANCES, REGULATIONS AND STANDARDS (LORS)

The following LORS were established to protect against the impacts of the noted criteria pollutants and the air toxics-related impacts of specific concern in this analysis.

FEDERAL

The Clean Air Act of 1970 (42 U.S.C., section 7401 et seq.)

This section of the act required establishment of the previously noted ambient air quality standards necessary to protect the public against effects in humans and the general environment. These standards were established by the United States Environmental

Protection Agency (EPA) for the major criteria pollutants: nitrogen oxides (NOx), ozone, sulfur dioxide, carbon monoxide, sulfates, lead, and particulate matter with a diameter of 10 micron or less (PM10).

The Clean Air Act of 1970 (42 U.S.C., section 7412)

This section requires new sources, which emit more than 10 tons per year of air toxics or any combination of air toxics, to apply the Maximum Achievable Control Technology (MACT).

STATE

California Health and Safety Code section 39606

This section of the code requires the California Air Resources Board (ARB) to establish California's ambient air quality standards to reflect the California-specific conditions influencing its air quality. Such standards have been established by the ARB for ozone, carbon monoxide, sulfur dioxide, PM10, lead, hydrogen sulfide, vinyl chloride and nitrogen dioxide. The California standards are listed together with the corresponding federal standards in the **Air Quality** section.

California Health and Safety Code section 41700

This section of the code states that "[n]o person shall discharge from any source whatsoever such quantities of air contaminants or other material which cause injury, detriment, nuisance, or annoyance to any considerable number of persons or to the public, or which endanger the comfort, repose, health, or safety of any such persons or the public, or which cause or have a natural tendency to cause injury or damage business or property."

California Health and Safety Code section 39650 et seq.

This section of the code mandates that the California Environmental Protection Agency (Cal-EPA) establish safe exposure limits for toxic, non-criteria air pollutants and identify the best available methods for controlling their emission. These laws also require that the new source review rules for each Air District include regulations establishing procedures for controlling the emission of these pollutants. The toxic emissions from natural gas combustion are listed in ARB's Toxic Emissions Factors (CATEF) database for natural gas-fired combustion turbines to allow for uniform assessment as emitted from combustion and non-combustion sources in the state. Cal-EPA has developed specific cancer potency estimates for assessing any cancer risk that these air toxics may pose at specific exposure levels. For toxic air pollutants that do not cause cancer, Cal-EPA established specific no-effects levels (known as reference exposure levels or RELs) for assessing the likelihood of producing health effects at specific exposure levels. Such health effects would be considered significant only when exposure exceeds these reference levels. Staff uses these Cal-EPA potency estimates and reference exposure values in its health risk analyses.

HEALTH AND SAFETY CODE SECTION 44300 ET SEQ.

This section of the code requires facilities, which emit large quantities of criteria pollutants and any amount of non-criteria pollutants to provide the local air district an inventory of toxic emissions. Operators of such facilities may also be required to

prepare a quantitative health risk assessment to address the potential health risks involved. The ARB ensures statewide implementation of these requirements through the state's Air Districts.

LOCAL

Bay Area Air Quality Management District Rule 2-1-316

This rule specifies the procedures necessary to minimize the emission of air toxics from specific sources as required by the Health and Safety Code section 44300.

Bay Area Air Quality Management District Regulation 1, Section 301, "Public Nuisance" (Amended 10/98).

Requirements of this regulation allow for implementation of the emission control measures necessary for compliance with provisions of the Health and Safety Code, section 41700.

SETTING

As noted by the applicant – Calpine, doing business as East Altamont Energy Center LLC – the proposed project site is a 40-acre portion of a 174-acre land parcel located in the far eastern corner of Alameda County. This site is approximately 8 miles northwest of the City of Tracy, 12 miles east of Livermore, 5 miles south of Byron, and less than 1 mile from the San Joaquin County border (EAEC 2001a, pages 8.6-1 and 12-1). This area of rural Alameda County is sparsely populated, as it is zoned for agriculture, electric utility corridors (such as substations, transmission lines, and wind farms), highways, recreation uses, and water management projects, with the actual project site currently used for agriculture.

Few residences are located in the vicinity of the site. The applicant indicated, and staff verified, that there is one "sensitive receptor" within a 3-mile radius of the project site (EAEC 2001a, pages 8.4-8, 8.12-1). This 3-mile radius is the area staff recognizes as potentially significant for the pollutant exposures of concern in this analysis. A sensitive receptor, for purposes of a public health analysis, is an establishment that houses sensitive individuals (e.g., children, the elderly, and individuals with respiratory diseases), such as a school, hospital, a daycare facility, or a nursing home. The sensitive receptor in this case is an elementary school (Mountain House School), located about 1 mile from the site. When there are many sensitive receptor locations in a project area, the probability of health complaints increases. However, staff holds all projects to the same health standards whether proposed for a major population center or a sparsely populated area.

The health effects of the air toxics of specific concern in this analysis are assessed individually by staff according to their potential to induce cancer or effects other than cancer. Staff would not recommend certification if any potential health impacts were determined to be significant as discussed below.

METHOD FOR ASSESSING THE CANCER AND NON-CANCER IMPACTS OF TOXIC AIR POLLUTANTS

Any air toxics-related health risks from operating the proposed EAEC and similar projects would mainly be associated with emissions from natural gas-fired combustion turbines, auxiliary burners, cooling towers, and auxiliary equipment such as diesel-fueled emergency generators and fire pumps. For the surrounding population, the risk of cancer or non-cancer effects is assessed from exposure estimates obtained from dispersion modeling. According to present knowledge, cancer begins with specific impacts at the genetic level, suggesting a specific (if theoretical) risk from every exposure to a carcinogen. The aim of present regulations is to eliminate all such exposures to the extent feasible for the source in question. This non-threshold concept is recognized as sharply contrasting with assumptions about non-cancer effects, which are assumed to result only from exposure above specific levels, meaning that significant health impacts would be prevented by maintaining exposures below the applicable exposure standards.

The procedure used for assessing such cancer and non-cancer impacts is known as a health risk assessment, which consists of the following steps:

Hazard identification – each pollutant of concern is identified along with possible health effects;

Dose-response assessment – the relationship between the magnitude of exposure and the probability of effects is established;

Exposure assessment – the possible extent of pollutant exposures from a project is established for all possible pathways by dispersion modeling; and

Risk characterization – the nature and the magnitude of the possible human health risk is assessed.

Health Effects Assessed

The risk assessment process addresses three categories of health impacts: acute (short-term) health effects, chronic (long-term) non-cancer effects, and cancer risk (also long-term).

Acute health effects result from short-term (1-hour) exposure to relatively high concentrations of pollutants, such as might occur in the event of an accidental spill. Acute effects are temporary in nature, and include symptoms such as irritation of the eyes, skin, and respiratory tract.

Chronic health effects are those which arise from long term exposure to lower concentrations of pollutants. The exposure period is considered to be greater than 12 percent of a lifetime of seventy years. Thus, human exposures of greater than eight years are considered chronic exposures. Chronic health effects include diseases such as cancer, reduced lung function and heart disease.

Estimating The Risk Of Non-Cancer Effects

The method used by regulatory agencies to quantify the likelihood of acute or chronic impacts of air toxics is the hazard index method. In the current assessment approach, a

hazard index is calculated as a numerical representation of the likelihood of significant health impacts at the exposure levels expected for the source being considered. This index is calculated by dividing the exposure estimate by the applicable reference exposure level (REL). After calculating the hazard indices for the individual pollutants, these indices are added together for all those that affect the same part of the body or target organ, to obtain a total hazard index for the source. Total hazard indices of 1.0 or less are regarded as indicating an insignificant addition to the non-cancer effects being considered. An index of more than 1.0 would reflect a potential for significant impacts.

Estimating The Risk Of Cancer

The risk of cancer is assumed to increase with duration of exposure, meaning for example, that the risk from longer exposures to carcinogens would be higher than the risk from shorter exposures. Theoretically, however, a single exposure to a carcinogen can cause cancer. Therefore, cancer is considered to be a more sensitive measure of potential adverse health effects than non-cancer risks.

For any source of specific concern, the risk of operations-related cancer is obtained by multiplying the exposure estimate by the potency factors for the individual carcinogens to be emitted. These potency factors are numerical values conservatively established to represent the cancer-causing potential of one carcinogen as compared to the others. After calculating these individual risk values, they are added together to obtain the total incremental cancer risk estimate from operating the project over a period conservatively assumed to span the 70-year lifetime of the average individual. Given the conservative nature of this risk calculation process, these numerical estimates are regarded as only representing the upper bounds on the project-related cancer risk at issue. The actual risk will likely be lower and could indeed be zero. The significance of these estimates as indicators of a real cancer hazard is assessed according to specific evaluative criteria as discussed below.

STAFF'S SIGNIFICANCE CRITERIA

Various state and federal agencies specify different cancer risk levels as levels of significance. For example, a risk of 10 in a million is mostly considered significant under the Air Toxics "Hot Spots" (AB 2588) and the Proposition 65 programs and, therefore, used as a threshold for public notification in cases of air toxics emissions from existing sources.

In the current regulatory practice, most health risk assessments are conducted in two phases. In the first phase (which is the screening phase), risk calculations are made using conservative, simplifying assumptions, which tend to overestimate rather than underestimate the risk. If the estimate from this screening-level analysis is below 10 in a million, staff regards the suggested cancer risk as insignificant and not warranting further analysis for specific action. If the estimate is more than 10 in a million, a more refined analysis (using more situation-specific assumptions) might be necessary to assess the need for mitigation. In such a refined analysis, staff would recommend specific mitigation only when the risk estimate is 10 in a million or more. This limit-based regulatory approach is intended to reduce the rate of addition to the high (1 in 4, or 250,000 in a million) background cancer risk of the average individual. While the causes of some types of cancers are well known, the causes of most of human cancers

remain largely unknown. What has become increasingly clear to scientists, however, is that environmental pollutants are responsible for only a small fraction of human cancers in general. The South Coast Air Quality Management District (SCAQMD 2000, page 2) estimated this fraction as only about two percent of cancer cases.

For non-carcinogenic pollutants, staff considers significant health impacts to be unlikely when the total hazard index is 1.0 or less. If more than 1.0, staff regards the related emissions as potentially significant from an environmental health perspective but would recommend specific mitigation only after considering the uncertainties in the assessment process.

IMPACTS

POTENTIAL IMPACTS OF PROJECT'S NON-CRITERIA POLLUTANTS

The health impacts of EAEC's air toxics of specific concern in this analysis can be assessed separately as construction-phase impacts and operational-phase impacts.

Construction Phase Impacts

Possible construction-phase health impacts, as noted by the applicant (EAEC 2001a, Appendix 8.1E), are those from human exposure to (a) the windblown dust from site excavation, and grading, and (b) emissions from construction-related equipment. The dust-related impacts may derive from exposure to the dust itself as PM10, or exposure to the toxic contaminants adsorbed on to it. Specific conditions of certification are proposed in the **Waste Management** section to prevent worker or public exposure to soil-bound contaminants. If these conditions are implemented, the only construction-related PM10 impacts of potential significance would derive from possible PM10 impacts as a criteria pollutant. As mentioned earlier, the potential for significant impacts arising from criteria pollutants is assessed in the **Air Quality** section.

Exhaust from diesel-fueled construction equipment has been established as a potent human carcinogen. Thus construction-related emission levels should be regarded as possibly adding to the carcinogenic risk of specific concern in this analysis. The maximum cancer risk from the use of diesel-fueled equipment for EAEC's construction was conservatively calculated by the applicant (EAEC 2001q) to be 11 in a million for the maximally exposed individual located near the project property line. As noted by the applicant, this screening-level calculation was made without adjusting for the ARB-noted reduction in PM10 that results from the use of low-sulfur fuel (which is proposed for the project). Adjusting for such reduction would yield a maximum risk of 8.25 in a million, which would be much lower at the nearest residences in this sparsely populated area. Such a screening-level risk estimate is not considered by staff as warranting more mitigation than specified in the applicant's Construction Mitigation Plan (EAEC 2001a, Appendix 8.1E). The implementing condition of certification is specified as **AQC-2** in the **Air Quality** section. Staff considers these conditions as adequate for preventing the cancer and non-cancer risks.

Operational Impacts

As noted in a publication by the South Coast AQMD (SCAQMD 2000, page 6), one property that distinguishes the air toxics of concern in this analysis from the criteria pollutants is that the impacts from air toxics tend to be highest in close proximity to the source and quickly drop off with distance. This means that the levels of EAEC's air toxics would be highest in the immediate area and would decrease rapidly with distance. One main focus of this analysis is to establish whether or not such exposures would be at levels of possible health significance as established using existing assessment methods.

The applicant's estimates of the EAEC's potential contribution to the area's carcinogenic and non-carcinogenic pollutants were obtained from a screening-level health risk assessment conducted according to procedures specified in the 1993 California Air Pollution Control Officer's Association (CAPCOA) guidelines. The results from this assessment were provided to staff along with documentation of the assumptions used (EAEC 2001a, pages 8.1-42 through 8.1-44, and pages 8.6-4 through 8.6-8). This documentation included:

Pollutants considered;

Emission levels assumed for the pollutants involved;

Dispersion modeling used to estimate potential exposure levels;

Exposure pathways considered;

The cancer risk estimation process;

Hazard index calculation; and

Characterization of project-related risk estimates.

Staff has found these assumptions to be acceptable and has validated the applicant's findings with regard to the numerical public health risk estimates expressed either in terms of the hazard index for each non-carcinogenic pollutant, or a cancer risk for estimated levels of the carcinogenic pollutants. These analyses were conducted to establish the maximum potential for acute and chronic effects on body systems such as the liver, central nervous system, the immune system, kidneys, the reproductive system, the skin and the respiratory system.

The following noncriteria pollutants were considered in this screening-level analysis with respect to non-cancer effects from the inhalation: ammonia from the use of the selective catalytic reduction (SCR) system for NOx control, acetaldehyde, acrolein, arsenic, benzene, chromium, copper, ethylbenzene, formaldehyde, hexane, lead, mercury, naphthalene, nickel, polycyclic aromatic hydrocarbons (PAHs), propylene oxide, silver, toluene, xylene, zinc, and 1,3-butadiene. The following were considered with regard to a possible cancer risk: acetaldehyde, arsenic, benzene, cadmium, chromium, formaldehyde, PAHs and propylene oxide, and 1,3-butadiene.

A maximum chronic hazard index of 0.086 was calculated for the maximally exposed individual, with an index of 0.14 similarly calculated for acute effects. These values are

well below staff's significance criteria, suggesting that these pollutants are unlikely to pose a significant risk of chronic or acute health effects anywhere in the project area.

The highest cancer risk was calculated as 0.96 in one million for all the project-related sources (gas turbines, auxiliary boiler, cooling tower, emergency generator, and fire engine), with the emergency pump responsible for approximately 0.9 in a million of this risk.

The relative contributions of the project's sources of the considered carcinogens are listed below:

Project Source	Potential Contribution to Total Cancer Risk
Gas turbines	0.00035 in a million
Auxiliary boiler	0.0475 in a million
Cooling tower	0.0000286 in a million
Emergency generator	0.0149 in a million
Fire pump engine	0.895 in a million
Total Cancer Risk	0.96 in a million

The conservatism in the employed risk calculation method is reflected by the fact that (a) the individual considered is conservatively assumed to be exposed at the highest possible levels to all the carcinogenic pollutants from the project for a 70-year lifetime, (b) all the carcinogens are assumed to be equally potent in humans and experimental animals, even when their cancer-inducing abilities have not been established in humans, and (c) humans are assumed to be as susceptible as the most sensitive experimental animal, despite knowledge that such cancer potencies often differ between humans and experimental animals. Only a relatively few of the many environmental chemicals identified so far as capable of inducing cancer in animals have been shown to cause cancer in humans.

CUMULATIVE IMPACTS OF AREA AIR TOXICS

When toxic pollutants are emitted from multiple sources within a given area, the cumulative, or additive, impacts of such emissions could, in concept, lead to significant health impacts within the population, even when such pollutants are emitted at insignificant levels from the individual sources involved. Analyses of such emissions have shown, however, that the peak impacts of such toxic pollutants are normally localized within relatively short distances from the source. Given the low cancer and non-cancer risks from all of EAEC's toxic emissions, coupled with the lack of other nearby toxic sources, staff has determined that the project will not contribute significantly to any area toxic exposure in a cumulative nature. The cumulative impacts of operational-phase criteria pollutants were assessed in the **Air Quality** section in establishing the potential extent of the needed emission offsets.

ENVIRONMENTAL JUSTICE

As noted in the **Socioeconomics** section, there are a few pockets of predominantly minority populations in the impact area of the proposed EAEC. The presence of such predominantly minority populations points to the possibility of environmental injustice in

human exposures to the project's air pollutants. Since (a) environmental injustice is encountered in cases of pollutant emissions at levels of potential health significance and (b) the health risk from the project's operations were established as potentially insignificant, staff has determined that the project operation will not result in a disproportionate adverse impact on minority or low-income populations.

RESPONSE TO PUBLIC AND AGENCY COMMENTS

JHS-1 *The commenter's concern includes the potential health risk from exposure to the pollutants from the proposed facility.*

Response. As noted in this analysis, the types of pollutants that are addressed in this Public Health analysis are unlikely (at potentially emitted levels) to pose a significant health risk to anyone in the project area.

FACILITY CLOSURE

As noted in the introduction section, the toxic pollutants of primary concern in this analysis are those from routine operation of the proposed project. During temporary or permanent closure, the main concern would be over the non-routine releases of hazardous materials or wastes on site. Such releases are discussed respectively in the **Hazardous Materials** and **Waste Management** sections. Since project operations would be stopped during forced temporary closures, any hazardous releases would not be in significant amounts. During permanent closure, the only emissions of potential significance would derive from demolition or dismantling activities and the equipment used. Such emissions would be subject to controls according to requirements in conditions adopted by the Energy Commission after a closure plan is received from the project owner.

CONCLUSIONS AND RECOMMENDATIONS

Staff has determined that the toxic air emissions from the operation of the proposed natural gas-burning EAEC and its auxiliary equipment are at levels that do not require mitigation beyond that already proposed by the applicant. The conditions for ensuring compliance with all applicable air quality standards are specified in the **Air Quality** section for the area's problem criteria pollutants.

The potential impacts from construction-related toxic exposures would be minimized through compliance with related conditions in the **Air Quality**, and **Waste Management** sections. Since these conditions are intended as protection against health impacts, additional conditions of certification are considered unnecessary in this **Public Health** section.

REFERENCES

BAAQMD (Bay Area Air Quality Management District) 1999. Toxic Air Contaminant Control Program. Annual Report, 1999.

ARB (California Air Resources Board) 1996. California Toxic Emissions Factors (CATEF) Database for Natural Gas-Fired Combustion Turbine Cogeneration, 1996.

CAPCOA (California Air Pollution Control Officers Association) 1993. Air Toxics "Hot Spots" Program, Revised 1992 Risk Assessment Guidelines. Prepared by the Toxics Committee, October, 1993.

EAEC (East Altamont Energy Center) 2001a. Application for Certification, Volumes 1& Appendices. East Altamont Energy Center (01-AFC-4). Dated March 20, 2001 and docketed March 29, 2001.

EAEC (East Altamont Energy Center) 2001q. Applicant's response to informal comments submitted by ARB, docketed August 28, 2001.

SCAQMD (South Coast Air Quality Management District) 2000. An Air Toxics Control Plan for the Next Ten Years. March 2000. South Coast Air Quality Management District publication, 2002.

ATTACHMENT A - CRITERIA POLLUTANTS

OZONE (O_3)

Ozone is not directly emitted from specific sources but is formed when reactive organic compounds (VOCs) interact with nitrogen oxides in the presence of sunlight. Heat speeds up the reaction, typically leading to higher concentrations in the relatively hot summer months. Ozone is a colorless, reactive gas with oxidative properties that allow for tissue damage in the exposed individual. The effects of such damage could be experienced as respiratory irritation that could interfere with normal respiratory function. Ozone can also damage plants and other materials susceptible to oxidative damage.

The U.S. EPA revised its federal ozone standard on July 18, 1997 (62 Fed. Reg. 38856), based on health studies that had became available since the standard was last revised in 1979. These new studies showed that adverse health effects could occur at ambient concentrations much lower than reflected in the previous standard, which was based on acute health effects experienced during heavy exercise. In proposing the new standard, the EPA identified specific health effects known to have been caused by short-term exposures (of one to three hours) and prolonged exposure (of six to eight hours) (61 Fed. Reg. 65719). However, a 1999 federal court ruling blocked implementation of the ozone 8-hour standard, which is yet to be implemented.

Acute health effects from short-term exposures include a transient reduction in pulmonary function, and transient respiratory symptoms including cough, throat irritation, chest pain, nausea, and shortness of breath with associated effects on exercise performance. Other health effects of short-term or prolonged O_3 exposures include increased airway responsiveness (which predisposes the individual to bronchoconstriction induced by external stimuli such as pollen and dust), susceptibility to respiratory infection (through impairment of lung defense mechanisms), increased hospital admissions and emergency room visits, and transient pulmonary inflammation.

Generally, groups considered especially sensitive to the effects of air pollution include persons with existing respiratory diseases, children, pregnant women, and the elderly. However, controlled exposure data on people in clinical settings have indicated that the population at greatest risk of acute effects from ozone exposures as children and adults engaged in physical exercise. Children are most at risk because they are active outside, playing and exercising, during summer when ozone levels are highest. Adults who are outdoors and engaging in heavy exertion in the summer months are also among the individuals most at risk. This happens because such exertion increases the amount of O_3 entering the airways and can cause O_3 to penetrate to peripheral regions of the lung where lung tissue is more likely to be damaged. These individuals, as well as those with respiratory illnesses, such as asthma, can experience a reduction in lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during periods of moderate exertion.

CARBON MONOXIDE (CO)

Carbon monoxide is a colorless, odorless gas, which is a product of inefficient combustion. It does not persist in the atmosphere, being quickly converted to carbon dioxide. However, it can reach high levels in localized areas, or "hot spots".

CO reduces the oxygen carrying capacity of the blood, thereby disrupting the delivery of oxygen to the body's organs and tissues. Persons sensitive to the effects of carbon monoxide include those whose oxygen supply or delivery is already compromised. Thus, groups potentially at risk to carbon monoxide exposure include persons with coronary artery disease, congestive heart failure, obstructive lung disease, vascular disease, and anemia, the elderly, newborn infants, and fetuses (CARB 1989, p. 9). In particular, people with coronary artery disease were found to be especially at risk from carbon monoxide exposure (CARB 1989, p. 9). Tests conducted on patients with confirmed coronary artery disease indicated that exposure to low levels of carbon monoxide during exercise can produce significant cardiac effects. These effects include chest pain (angina) and electrocardiographic changes indicative of effects on the heart muscle (CARB 1989, p. 6). Such changes can limit the ability of patients with coronary artery disease to exert themselves even moderately. Therefore, the statewide carbon monoxide one-hour and eight-hour standards were adopted in part to prevent aggravation of chest pain. Additionally, however, the standards are intended to prevent decreased exercise tolerance in persons with peripheral vascular disease and lung disease, impaired central nervous system functions, and effects on the fetus (Cal. Code Regs. Tit. 17, 70200).

PARTICULATE MATTER (PM)

Particulate matter is a generic term for particles of various substances, which occur as either liquid droplets or small solids of a wide range of sizes. Particles with the most potential to adversely affect human health are those less than 10 micrometers (millionths of a meter) in diameter (or PM10), which may be inhaled and deposited within the deep portions of the lung (PM10). PM may originate from anthropogenic or natural sources such as stationary or mobile combustion sources or windblown dust. Particles may be emitted directly to the atmosphere or result from the physical and chemical transformation of gaseous emissions such as sulfur oxides, nitrogen oxides, and volatile organic compounds. PM10 may be made up of elements such as carbon, lead, and nickel; compounds such as nitrates, organics, and sulfates; and complex mixtures such as diesel exhaust and soil fragments. The size, chemical composition, and concentration of ambient PM10 can vary considerably from area to area and from season to season within the same area.

PM10 can be grouped into two general sizes of particles, fine and coarse, which differ in formation mechanisms, chemical composition, sources, and potential health effects. Fine-mode particles are those with a diameter of 2.5 micrometers or less (PM2.5), while the coarse-mode fraction of PM consists of particles ranging from 10 micrometers down to 2.5 micrometers in diameter. A 1999 federal court ruling blocked implementation of these standards, which is yet to be implemented.

PM2.5 is derived both from combustion by-products, which have volatilized and condensed to form primary PM2.5 and from precursor gases reacting in the atmosphere

to form secondary PM2.5. Components include nitrates, organic compounds, sulfates, ammonium compounds, and trace elements (including metals) as well as elemental carbon such as soot. Major sources of PM2.5 are fossil fuel combustion by electric utilities, industry and motor vehicles, vegetation burning, and the smelting or other processing of metals. Dry deposition of fine mode particles is slow allowing such particles to often exist for long periods of time (of from days to weeks) in the atmosphere and travel hundreds to thousands of kilometers. They tend to be uniformly distributed over urban areas and larger regions and are removed from the atmosphere primarily by forming cloud droplets and falling out within raindrops.

Coarse-mode PM10 is formed by crushing, grinding, and abrasion of surfaces, and in the course of reducing large pieces of materials to smaller pieces. Coarse particles consist mainly of soil dust containing oxides of silicon, aluminum, calcium, and iron; as well as fly ash, particles from tires, pollen, spores, and plant and insect fragments. Coarse particles normally have shorter lifetimes (minutes to hours) and only travel over short distances (of less than tens of kilometers). They tend to be unevenly distributed across urban areas and have more localized effects than the finer particles.

The health effects of PM10 from any given source usually depend on the toxicity of its constituent pollutants. The size of the inhaled material usually determines where in the respiratory it is deposited. Coarse particles are deposited most readily in the nose and throat area while the finer particles are more likely to be deposited within the bronchial tubes and air sacs, with the greatest percentage deposited in the air sacs. Particles deposited in the air sacs are removed more slowly by the body's particulate defense system than those deposited in the nose and throat area. Deposition in the air sacs allows for the longer residence time necessary for impacts of potential health significance.

Many epidemiological studies have shown exposure to particulate matter as capable of a variety of health effects, including premature death, aggravation of respiratory and cardiovascular disease, changes in lung function and increases in existing respiratory symptoms, effects on lung tissue structure, and impacts on the body's respiratory defense mechanisms. The underlying biological mechanisms are still poorly understood. Based on their review of a number of these epidemiological studies (as published after 1987 when the federal standards were last revised), together with suggestion of PM2.5 concentrations as a more reliable surrogate for the health impacts of the finer fraction of PM than PM10, EPA concluded that the then-current standards were not sufficiently stringent to protect against significant effects in exposed humans. Therefore, federal PM standards were revised on July 18, 1997 (62 Fed. Reg. 38652) to add new, annual and 24-hour PM2.5 standards to the existing annual and 24-hour PM10 standards. Taken together, these new standards were meant to provide additional protection against a wide range of PM-related health effects, including premature death, increased hospital admissions and emergency room visits, primarily among sensitive individuals such as the elderly, children and individuals with cardiopulmonary diseases such as asthma. Other impacts include decreased lung function (particularly in children and asthmatics), and alterations in lung tissue and structure.

California has 24-hour and annual standards for only PM10 are based on symptoms observed at the lowest concentrations used in human studies (CARB 1982, pp. 81,84). These studies were aimed at establishing the PM10 levels capable of inducing asthma, premature death and bronchitis-related symptoms. They were set to protect against such impacts in the general population as well as sensitive individuals such as patients with respiratory disease, declines in pulmonary function, especially as related to children (Cal. Code Regs. Tit. 17, 70200). These standard was set to be more stringent than the federal standard, which the ARB regards as inadequate for the protection desired (CARB 1991, p. 26).

The annual standard is based on studies showing long-term exposure to PM10 as capable of decreasing breathing capability and increasing respiratory illnesses among susceptible individuals, especially children (CARB 1991, p. 25). The annual standard is also set to also accommodate the need for protection against any carcinogenic effects of PM10 (CARB 1982, p. 84).

NITROGEN DIOXIDE (NO_2)

Nitrogen dioxide is formed either directly or indirectly when oxygen and nitrogen in the air combine together during the combustion. It is a relatively insoluble gas, which can penetrate deep into the lungs, its principal site of toxicity. Its toxicity is thought to be due to its capacity to initiate free radical-mediated reactions while oxidizing cellular proteins and other biomolecules (CARB 1992, Appendix A, p. 4).

Sub lethal exposures in animals usually produce inflammations and varying degrees of tissue injury characteristic of oxidant damage (Evans in CARB 1992, Appendix A, and p 5). The changes produced by low-level acute or sub chronic exposures appear to be reversible when the animal study subject is allowed to recover in clean air.

Health effects of particular concern in relation to low-level nitrogen dioxide exposure include: (1) effects of acute exposure on some asthmatics and possibly on some persons with chronic bronchitis, (2) effects on respiratory tract defenses against infection, (3) effects on the immune system, (4) initiation or facilitation of the development of chronic lung disease, and (5) interaction with other pollutants (CARB 1992, Appendix A, p. 5).

Several groups, which may be especially susceptible to nitrogen dioxide-related health effects have been identified from human studies (CARB 1992, Appendix A, and p. 3). These include asthmatics, persons with chronic bronchitis, infants and young children, cystic fibrosis and cancer patients, people with immune deficiencies, and the elderly.

Studies involving brief, controlled exposures on sensitive individuals have shown an increase in bronchial reactivity or airway responsiveness of some asthmatics, as well as decreased lung function in some patients with chronic obstructive lung disease (CARB 1992, Appendix A, p. 2). In general, bronchial hyper reactivity (an increased tendency of the airways to constrict) is markedly greater in asthmatics than in non-asthmatics upon exposure to initiating respiratory irritants (CARB 1992a, p. 107). At exposure concentrations of specific relevance to the current one-hour ambient standard, there

appears to be little, if any, effect on respiratory symptoms of asthmatics (CARB 1992a, p. 108).

SULFUR DIOXIDE (SO_2)

Sulfur dioxide is formed when any sulfur-containing fuel is burned. SO_2 is highly soluble and consequently absorbed in the moist passages of the upper respiratory system. Exposure to sulfur dioxide can lead to changes in lung cell structure and function that adversely affect a major lung defense mechanism known as muco-ciliary transport. This mechanism functions by trapping particles in mucus in the lung and sweeping them out via the cilia (fine hair-like structures) also in the lung. Slowed mucociliary transport is frequently associated with chronic bronchitis.

Exposure to sulfur dioxide can produce both short- and long-term health effects. Therefore, California has established sulfur dioxide standards to reflect both short- and long-term exposure concerns. Based on controlled exposure studies of human volunteers, investigators have found that asthmatics comprise the group most susceptible to adverse health effects from exposure to sulfur dioxide (CARB 1994, p. V-1).

The primary short-term effect is bronchoconstriction, a narrowing of the airways, which results in labored breathing, wheezing, and coughing. The short-term (one-hour) standard is based on bronchoconstriction and associated symptoms (such as wheezing and shortness of breath) in asthmatics and is designed to protect against adverse effects from five to ten minute exposures. In the opinion of the California Office of Environmental Health Hazard Assessment, the short-term ambient standard is likely to afford adequate protection to asthmatics engaged in short periods of vigorous activity (CARB 1994, Appendix A, p. 16).

Longer-term exposure is associated with increased incidence of respiratory symptoms (such as coughing and wheezing) or respiratory disease, decreases in pulmonary function, and an increased risk of premature mortality (CARB 1991a, p. 12). The long-term (24-hour) standard is based upon increased incidence of respiratory disease and premature mortality. The standard includes a margin of safety based on epidemiological studies, which have shown adverse respiratory effects at levels slightly above the standard. Some of the studies indicate a sulfur dioxide threshold for effects, suggesting that no significant effects are expected from exposures to concentrations at the state standard (Ibid.).

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